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A Field Trial of a Parasitic Bronchitis Vaccine

BY

W. F. H. JARRETT, F. W. JENNINGS, B. MARTIN, W. I. M. McINTYRE, W. MULLIGAN, N. C. C. SHARP, and G. M. URQUHART

University of Glasgow Veterinary School

SUMMARY.—A small-scale field trial of a parasitic bronchitis vaccine is described. The vaccine was made by attenuating infective larvae by X-irradiation. A considerable degree of protection was conferred on vaccinates as judged by clinical and pathological findings, mortality, morbidity, faecal larval counts, and worm burdens. The application of this vaccination procedure in the field is discussed.

Introduction

It has been demonstrated (Jarrett *et al.*, 1957) that it is possible to immunise calves against *Dictyocaulus viviparus* in 3 different ways, *viz.* by injecting immune serum intraperitoneally, by using an adjuvanted whole worm vaccine intramuscularly, and by oral dosing with X-irradiated larvae.

On consideration of the epidemiology of parasitic bronchitis, it is obviously preferable to use a vaccine rather than a serum to control the disease and we decided to use the irradiated larval vaccine since it is cheaper to produce and gives a much higher degree of protection than the whole worm vaccine. In the experiments reported previously, the degree of immunity conferred by the vaccine was assessed quantitatively by vaccinating calves and subsequently challenging them with a single dose of normal infective larvae; these calves were killed 30 days after challenge and the worms in their bronchi were counted.

In order to estimate the utility of vaccination as a field procedure it was necessary to expose vaccinated calves to natural challenge while simultaneously employing some quantitative standard of measuring resistance. This has been done in two ways:—

(i) Of 1,000 calves on commercial farms with a recent history of severe husk, 500 were vaccinated and 500 were left as controls; a clinical and faecal examination was carried out monthly on each calf.

(ii) A number of calves were infected artificially and were then allowed to graze on a pasture which was subsequently grazed by groups of vaccinated and control calves. These latter groups were examined 3 times weekly. The present report deals with the second of these experiments; the former will be reported later.

Materials and Methods

Forty-five Ayrshire bull calves were bought when 2 to 7 days old and were reared parasite-free by housing in individual isolation pens and feeding artificially. They were randomised into 3 groups of 15, *viz.* carriers, vaccinates, and controls.

Carriers. The carrier calves were divided into 2 sub-groups of 5 and 10, one lot being infected with 4,000 normal third-stage larvae 45 days after the other.

Vaccinates. Fifty days before being turned out to graze, the vaccinate group were each given 1,000 X-irradiated larvae (the computation of the roentgen dosage used will be discussed in a later paper).

Controls. These calves received no treatment of any kind before being turned out to graze.

The design of the experiment. Five carrier calves were put on to a 3-acre pasture for 40 days while their faeces contained large numbers of larvae. These were then taken off and the remaining carriers, vaccinates, and controls were allowed to graze the paddock. (The control group now numbered 12 since 3 had been disposed of by accident.)

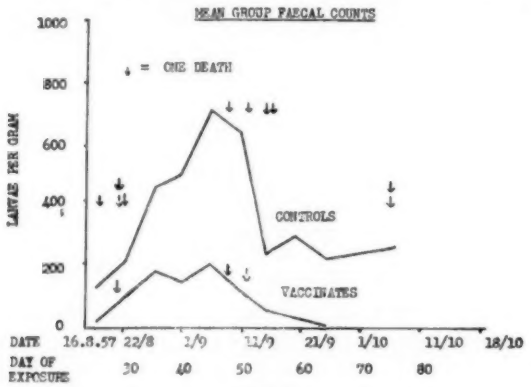
At a conservative estimate, 200,000,000 larvae were deposited on the pasture by the carriers giving an approximate concentration of 1,300 larvae per square foot; this was, of course, considerably augmented by

calves of the other groups which became infected during the experiment.

All of the calves were gathered together for clinical and faecal examinations 3 times weekly; the faeces were subjected to a modified McMaster technique.

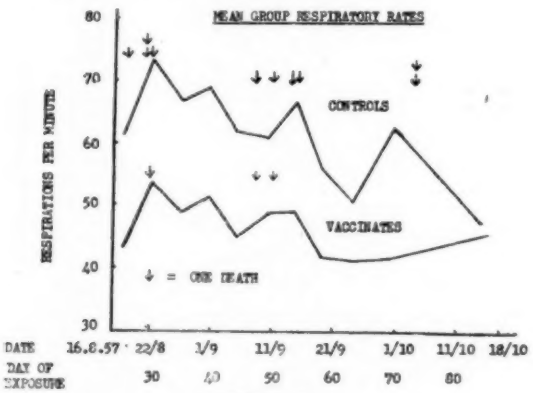
Results

The average faecal larval levels in the control and vaccinate groups are shown in Graph 1. The highest



GRAPH 1
MEAN FAECAL LARVAL LEVELS AND DAYS ON WHICH DEATHS OCCURRED IN VACCINATE AND CONTROL GROUPS

levels were reached at 44 days after grazing of the pasture began and the difference between the two groups is highly significant at this point. The difference would have been even greater had not four of the most highly infected controls died before this point. The successive deaths of controls after this time caused an unusually sharp drop in the average level of the group. The peak larval counts and the duration of larval counts in the two groups are shown in Tables I and II. The average respiratory rates of the two groups are given in Graph 2.



GRAPH 2
MEAN RESPIRATORY RATES IN VACCINATE AND CONTROL GROUPS

Ten of the 12 controls died between the 27th and 73rd day after being turned out; the specific day on which deaths occurred and the worm burdens are recorded in Table III. The mortality rate is 83 per cent. Three of the 15 vaccinates died, a mortality rate of 20 per cent.; details are given in Table III.

At autopsy and subsequent histological examination the lungs of calves dying during the experiment had typical lesions of parasitic bronchitis (Jarrett *et al.*, 1957); in addition the vaccinate which died 50 days after exposure showed widespread acute interstitial pneumonia.

TABLE I
NUMBER OF DAYS ON WHICH FAECES CONTAINED LARVAE AND PEAK LARVAL COUNTS ATTAINED IN CONTROL GROUP

Calf number	Days positive/days sampled	Highest count recorded
1	2/2	450
2	2/2	100
3	2/3	250
4	2/3	1,900
5	11/11	1,900
6	12/13	2,650
7	12/13	1,850
8	9/12	550
9	16/19	1,150
10	15/18	600
11	10/21	700
12	8/20	120

TABLE II
NUMBER OF DAYS ON WHICH FAECES CONTAINED LARVAE AND PEAK LARVAL COUNTS OBTAINED IN VACCINATE GROUP

Calf number	Days positive/days sampled	Highest count recorded
13	3/4	250
14	8/9	650
15	10/11	1,550
16	16/22	1,600
17	16/22	800
18	12/21	250
19	8/22	250
20	7/22	200
21	6/18	250
22	8/22	100
23	5/22	100
24	5/22	100
25	3/22	150
26	2/22	150
27	2/22	100

TABLE III
TIME OF DEATH AND WORM COUNT

Calf number	Group	Days after exposure on which death occurred	Worm count
1	Control	26	1,200
3	,,	29	930
2	,,	29	1,560
4	,,	30	4,670
5	,,	47	2,100
8	,,	50	910
6	,,	53	1,210
7	,,	54	2,250
9	,,	73	2,600
10	,,	73	3,000
13	Vaccinate	30	1,660
14	,,	47	840
15	,,	50	1,300

The survivors were killed when all faecal counts became negative. One vaccinate had 2 lungworms and the remainder none; one other had a small area of consolidation in a diaphragmatic lobe. Both of the controls showed some consolidation but neither was severe.

Discussion

In our previous experiments we used two variations in making attenuated vaccines, *viz.* the amount of X-irradiation to which infective larvae were subjected and the number of such larvae used to immunise each calf. It was found that both of these variations were reflected in the potency and safety of the vaccine.

If the irradiation was too great, the larvae failed to penetrate the host and no immunity resulted; if the roentgen dosage was too low, the larvae were not sufficiently attenuated, some reaching the lungs and there producing lesions which gave rise to coughing and increased respiratory rates in the calves. A suitable magnitude or irradiation was arrived at and the quantitative aspects of this have been discussed elsewhere (Jarrett *et al.*, in press).

Most of the experimental work was done using 4,000 larvae as the vaccinating dose and this proved to be 99 per cent. effective under certain conditions, *viz.* if calves were challenged with 4,000 normal infective larvae 50 days after vaccination and killed after a further 30 days the worm burdens were reduced from an average of 1,000 in controls to about 10.

This was a very acceptable degree of protection but it was felt that the vaccine dose for use in the field during early trials should be reduced to 1,000 larvae since if a failure of attenuation did occur, this number of larvae would not produce fatal disease. Experiments using single-dose artificial challenge showed that 1,000 larvae gave a reduced but still considerable degree of resistance to infection. This dose was used in the present experiment to assess its utility under field conditions of challenge.

The weather conditions during the course of this trial were very conducive to maintaining a high level of infective larvae on the pasture and it is unlikely that the levels produced by the 15 carriers, all severe cases of husk, are often obtained under commercial farming conditions. It is considered that this system effected a very rigorous challenge to any vaccine designed for field use.

Success of such a vaccine should be judged by three criteria: it must reduce or abolish mortality; it should diminish the number of larvae being deposited on the pasture so that after a few years any individual farm should have been largely cleared of the disease; and it should remove the need to house, and supply supplementary feeding to, susceptible animals during the grazing season.

The results leave little doubt that a very high degree of resistance was produced. Whether enough immunity would be produced by a vaccine of this strength to save all calves subjected to infection under field conditions is unknown but it would appear that the vaccine would be capable of considerably reducing mortality.

The reason for the apparent failure of the vaccine to protect three of the vaccinate group is unknown. Pathological examination of the lymph nodes and lungs failed to reveal the reactions usually seen in vaccinated calves.

On the second criterion of utility there is no doubt; the larval output of the controls was very significantly greater than the vaccinates both in numbers of larvae at any one time and in the length of time over which larvae were excreted.

The third criterion is difficult to assess in this trial because of the severe and prolonged nature of the challenge and the fact that calves, carriers, and controls were maintaining pasture levels which would not occur on a farm on which all calves were vaccinated. It was easily seen, however, a fortnight after the trial began that the vaccinates were in much better condition than the controls. This became less appreciable in the middle period but recovery in the vaccinated group was fairly rapid. The clear difference in the respiratory rates between controls and vaccinates shows the benefit conferred by vaccination.

When the surviving vaccinates were killed there were virtually no pulmonary lesions. Apart from the obvious point that the animals had been protected in great measure against the effects of husk, this demonstrated that the vaccinating dose of irradiated larvae had produced no permanent changes in the lungs.

It would appear from the experiment that this form of vaccination might have an important part to play in the control of husk in the field, both in reducing morbidity and mortality and in reducing pasture contamination with larvae on a farm. We think that the dose of vaccine (1,000 larvae) used in the above trial is probably sufficient to protect the great majority of animals at risk; but it is known that in the endemic areas such as south-west Scotland, some calves are exposed to very large infections (as many as 6,000 worms being found at autopsy), and these may require a greater degree of protection. For this purpose, we are exploring the use of double vaccination since it has been shown (Jarrett *et al.*, 1957) that an important factor in the immunity against *D. viviparus* is the time taken for the anamnestic or secondary response to develop. After an initial infection, the complement fixing antibody titre does not reach its maximum point until about 80 to 100 days; with subsequent re-infections this time is reduced to 10 to 14 days. The aim in double vaccination is to have an animal, when turned out to graze, capable of producing a high level of antibodies within a fortnight of ingesting infective larvae thus preventing the establishment of adults in the bronchi.

The results given by this series of experiments have led us to explore the use of attenuated larval vaccines in infestations by other species of worms and we are at present investigating *Fasciola hepatica* and *gigantica, Cysticercus bovis, Ascaris lumbricoides, Metastrongylus apri, D. filaria, Haemonchus contortus,* Nematodirus species, and Hookworms.

(*Concluded at foot of col. 1 overleaf*)

Hair Loss and Other Toxic Effects of Leucaenia glauca ("Jumbey")

BY

L. N. OWEN

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SUMMARY.—A study of the literature relating to the toxicity of Leucaenia glauca has been correlated with current observations made in the Bahamas.

Loss of hair in horses eating Leucaenia glauca is caused by the toxic amino acid mimosine. The long hair of the mane and tail is mainly affected. Ring formation occasionally occurs on the hooves. Similar hair loss occurs in pigs, and the effect on both horses and pigs is the same in the Bahamas as in Indonesia.

In many countries including Hawaii, Indonesia, and the Bahamas, Leucaenia glauca is a valuable forage plant, and it appears to have no toxic effects in ruminants. Native sheep in the Belgian Congo fed on locally grown Leucaenia glauca showed a fall of wool and developed a haemorrhagic cystitis.

Mimosine can be estimated by employing a colour reaction with ferric chloride. The toxicity of mimosine for rats is reduced following its subjection to moist heat or when ferrous sulphate is included in the ration.

Introduction

LEUCAENIA glauca is an arborescent legume belonging to the Mimosaceae. Its original habitat is Mexico but it is now found throughout the tropics. There is an extensive literature in Dutch, English, and French dealing with its properties and uses. This article reviews reports relating to its toxicity, and includes current observations made in the Bahamas.

The legume is known variously as "Jumbie Bean," "Jumbey," or "Jimbey" in the Bahamas, "Lamtoro" in Indonesia, and "Koa Haole" in Hawaii. It is well naturalised and widespread in the Bahama Islands, and in certain drier areas flourishes where little else will grow. The annual average rainfall in the Bahamas varies from 60 inches in the north at Green Turtle Cay, Abaco, to 28 inches in the south at Inagua. However, the extreme porosity of the limestone and coral rock means that many areas become very arid. The growth of Leucaenia flourishes following the May or June rains but in the drier winter months growth is arrested.

Soil *p*H appears to be unimportant as reports in the literature (Takahashi, 1952) state that it will grow in poor acid soils, while all the soils in the Bahamas are very alkaline.

The foliage is used extensively in the Pacific for feeding dairy and beef cattle, and is found useful as fodder in Australia in the dry season when no grass is available (Hill, 1957). In the Bahamas, sheep and goats often feed solely on Leucaenia during the long periods of drought which occur and, in spite of its known toxic effect in horses—notably the falling out of hair, it is still used occasionally to form an important source of food for this species because in severe drought there is little other food available.

The most valuable properties of this tropical and sub-tropical forage plant are its drought resistance, its ability to grow in poor soils, and its high percentage of protein.

Toxic Effects in Horses

Reference to the effects of Jumbey in horses was made by Morris in 1896, who stated that the loss of hair from the tail was called "cigar tail" by the inhabitants of Nassau in the Bahamas. He described hoof complications as appearing after a long period of feeding. Observation on loss of hair was also made by members of the Baltimore Geographical Expedition in their report on the Bahamas in 1905. They stated: "We also found in these islands specimens of the Jumbie Bean. This bean, when eaten by mules or horses, causes the hair of the mane and tail to fall out, giving a rather remarkable appearance to the animals who have been unfortunate enough as to have the bean as a diet." Mullenax has also noted the existence of this condition.

Loss of hair due to the feeding of Leucaenia glauca is still a well recognised condition in the Bahamas to-day, occurring relatively frequently. Emaciation and pathological conditions of the hooves, which are also of common occurrence, are in many cases associated with the ingestion of Leucaenia. Horses with loss of mane and tail hair are occasionally seen in the island of New Providence, but the percentage incidence is small because race-horse, surrey, and dray owners refuse to feed it. Similarly in the out-islands the incidence is not related to the quantity of Leucaenia available, but to the prejudices of the owners and the availability of other food. Thus in Long Island in 1956 only 6 out of 60 horses examined were affected, while in Eleuthera 16 out of 22 were affected, many severely. (See Figs. 1, 2, and 3.)

A Field Trial of a Parasitic Bronchitis Vaccine—*Concluded.*

Encouraging results have been obtained but it remains to be seen if parasites with different migration routes will be as susceptible to this form of attack as is *D. viviparus*.

Acknowledgments.—This work was supported by a grant from the Agricultural Research Council.

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JARRETT, W. F. H., JENNINGS, F. W., MCINTYRE, W. I. M., MULLIGAN, W., & URQUHART, G. M. (1957). *Vet. Rec.* **69**. 1,329.

NATURAL CASE OF LEUCAENIA GLAUCA TOXICITY IN A HORSE IN ELEUTHERA, 1957.

FIG. 1.—Loss of most of tail hair. FIG. 2.—Loss of most of mane hair. FIG. 3.—Ring formation on hooves.

The condition seen here varies from a slight loss of hair on the mane and tail to one where only a few hairs on the mane and the proximal part of the tail remain. Severe cases show in addition a patchy loss of hair below and above the knees and hocks and on the flanks and neck. Ring formation occasionally occurs on the hooves. There is a loss of condition and the owners of riding horses say that marked weakness occurs. After removal from a Leucaenia pasture, growth of hair of the original colour recurs after varying intervals of time, usually a month or more. In severely affected cases a normal appearance is not regained for 6 months. Horse owners say that the effect is more marked when the beans are fed than when leaves alone are given, an observation which is probably true as there is a higher percentage of the toxic factor, mimosine, in the beans than there is in the leaves.

In collaboration with Kostermans, Kraneveld, and Djaenoedin have performed feeding experiments on horses. In the case of one horse the feeding of dried seeds caused the hair to become less firmly attached to the skin and rubbing by the animal caused bald spots to appear. In the second animal this feeding caused part of the long hairs of the mane and tail to fall. Two experiments with dried foliage produced similar results.

The toxic amino acid, mimosine, first reported as occurring in Leucaenia glauca by Renz, and later successfully isolated by Kostermans, was then administered to further horses. Administration of 200 grammes in 4 days of a product consisting of 80 to 90 per cent. mimosine with sugar and chlorophyll, caused a horse to show the following effects: stomatitis, haemorrhagic enteritis, proctitis, oedema of the hind legs and genitals, acute laminitis followed by ring formation on all the hooves. A considerable part of the long hairs of the mane and tail were shed. Another experiment confirmed the toxic properties of mimosine. When smaller quantities were fed, loss of hair only with no other toxic signs occurred. Abouth a month after the end of the trial, the first regrowth of hairs on the bald patches appeared and it was the same colour as the original hair. It appears in some instances that horses may grow tolerant to the toxic agent, new hairs starting to grow on the bald patches in spite of continued feeding. Histological examination of the skin from the mane and tail of Leucaenia-fed horses showed only oedema of the corium.

Toxic Effects in Pigs

Morris (1896) stated that in pigs the action of Leucaenia went farther than in horses and that the animals became completely bald. Loss of hair in pigs has also been reported by workers in Hawaii. Kraneveld and Djaenoedin found that while some pigs refused to eat the plant, others became accustomed to it. Fifteen to 18 days after feeding the leaves, the bristles of the middle of the neck and back began to fall out or break off. Gradually the hair coat on the places mentioned became thin and then, despite continued feeding, new hair began to grow, and no case of complete baldness ever occurred. This regrowth of hair described for Indonesian pigs also often occurs in the native Bahamian pigs and may be connected with the time of the year and the growth of the plant.

Kostermans, however, fed a pig over a 3-week period with some 13 kg. of ripe Leucaenia seeds mixed with red rice, but there was no loss of hair. As Kraneveld and Djaenoedin remark, differences

in susceptibility between various breeds of pigs, or differences in the chemical composition of the plant could be a cause of these differing observations.

Toxicity in Ruminants

Branches and leaves of Leucaenia are often used as cattle and goat feed in Indonesia, and confirmatory trials in goats have established its non-toxicity in this species (Kraneveld & Djaenoedin, 1947). Holstein cows in Hawaii were found to consume 60 to 65 lb. of green Leucaenia glauca a day. Both lactation efficiency and cost per unit quantity of milk were slightly better when feeding Leucaenia than when Napier grass and concentrates were fed. However, Napier grass, when fertilised, yielded 75 tons an acre and Leucaenia only 25 tons, so that land values or availability become factors (Henke & Morita, 1954).

Hawaiian workers report that beef and dairy cattle show no toxic effects, and that this also applies to other ruminants. Exception occurred in rare cases when some loss of hair in the brush of the tails of cattle was observed. Feeding the legume too near milking time may impart a flavour to the milk.

No observable toxic effects directly attributable to Leucaenia occur in sheep and goats in the Bahamas, although many of these animals are in poor condition due to insufficient feed and to parasitic gastroenteritis. The legume is often the main food available and is readily eaten. Three experimental sheep fed solely on fresh cut leaves for 7 months maintained weight and condition, showed no wool or hair changes, and voided normal urine.

However, Damseaux in the Belgian Congo found that sheep would not readily eat the plant, and that the small amount which was eaten produced a fall of wool 10 to 14 days after the first ingestion. Voided urine was red and autopsy revealed a haemorrhagic cystitis. A repetition of the experiment produced similar results.

These striking effects may be due to a higher percentage of mimosine in the variety of plant introduced, or to a lack of natural or acquired resistance in the breed of sheep kept in the Congo. It is evident that the toxic principle is not destroyed in the rumen.

Toxicity in Other Animals

A report of hair loss in women of various ages in the Kingdom of Soemdang in Indonesia was ascribed to the fact that raw, unripe Lamtoro pods picked in the bush had been eaten. It was observed that even weeks after the falling out of hair the scalp was still somewhat oedematous, although a new growth of hair had already begun (Kraneveld & Djaenoedin, 1950).

Kostermans, using Leucaenia glauca grown in Indonesia, could produce no toxic effects in rats, doves, or in two apes. Similarly, Kraneveld and Djaenoedin report that 20 rabbits and 20 guinea-pigs remained normal after feeding 100 grammes of leaves daily for 2 months.

Hawaiian workers report, however, that where Leucaenia is ingested as the main forage, there is fall of hair and inhibition of growth, not only in horses, mules, donkeys, and pigs, but also in rats and rabbits. Four days after albino rats were fed Leucaenia glauca, small amounts of short hair were shed, and at the end of 14 weeks the animals were from 50 to nearly 100 per cent. bald. The males appeared to be affected more than the females. Food consumption was reduced and growth rate of these rats was less than half that of rats in a positive control group. Four animals developed cataracts. It appears that the growth rate is sharply reduced when the percentage of mimosine is raised beyond 0.75 per cent. of the ration (Matsumoto, Smith & Sherman, 1951).

Chemistry of Mimosine

Malmquist has shown that mimosine is present in Leucaenia glauca as an unbound amino acid. The correct structural formula of mimosine, β (N-3 hydroxypyridone-4) α amino proprionic acid, was first proposed by Wibaut and proved by Adams and Johnson with the synthesis of d.l. mimosine. The identity of mimosine isolated from Mimosa pudica with leucaenol or leucanine from Leucaenia glauca has been proved by Wibaut and Kleipool. A quantitative determination based on the colour reaction of mimosine with ferric chloride has been developed by Yoshida. A rapid colorimetric method for its determination devised by Matsumoto and Sherman utilises the ferric chloride colour reaction, after removing plant pigments by absorption with activated carbon.

Yoshida added ferrous sulphate to a ration containing mimosine and obtained less toxic effects, an increase in faecal mimosine, and a decrease in urinary mimosine when the ration was fed to rats. Matsumoto, Smith and Sherman (1951) found that the mimosine content of leaves and seeds stored at elevated temperatures is decreased. This effect was most pronounced and rapid when the temperature was over 70° C. in the presence of moisture, but did not occur when dry leaves were used. Fresh heat-treated material proved to be less toxic to rats than unheated leaves and seeds.

Acknowledgments.—My thanks are due to Miss M. M. Raymer of the Royal College Memorial Library who obtained references and kindly arranged translations from articles written in Dutch, and to Mr. B. D. Owen, B.SC., F.R.I.C., of the Murphy Chemical Co., Wheathampstead, Herts, who obtained other references for me.

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An Outbreak of Rinderpest Involving Cattle and Sheep

BY

R. H. JOHNSON

Federal Department of Veterinary Research, Vom, Nigeria

SUMMARY.—1. *An outbreak of rinderpest, originating in cattle, and affecting contact sheep is described.*

2. *The possible reasons for the irregular behaviour of the rinderpest virus in sheep are discussed.*

Introduction

ALTHOUGH rinderpest is widely stated to cause a natural infection in sheep and goats, recent reports of such infection are rare; Curasson (1942), Plowright (1952), and Beaton (1954). Reports of rinderpest epizootics involving sheep and goats are numerous in the older literature, and are reviewed by Curasson (1942) and Scott (1955), the latter author observing, however, that such reports have been confined for the last ninety years to Indian sheep and goats. Thus, Ware, cited by Jackson and Cabot (1930), refers to occasional foci in the Mysore province of India in which the disease in sheep and goats is relatively common, and D'Costa and Singh (1933), describe rinderpest in goats. Srikantaiah (1934), in discussing rinderpest vaccine reactions in sheep, makes reference to natural cases of the disease in these animals. D'Costa (1936), also notes cases of rinderpest affecting cattle and sheep, and Bawa (1940) and Chowdry (1941) describe outbreaks of rinderpest in sheep and goats. Similarly, Lall (1947), in discussing rinderpest vaccination of sheep and goats, refers to the natural disease in these animals, and Dhanda and Manjrekar (1952), review the Indian literature and describe an outbreak involving sheep and goats. Mohan and Bahl (1953) report rinderpest in goats associated with cutaneous lesions. Rinderpest in imported Indian goats in Singapore, is reported by Whitworth (1936), in Ceylon by Crawford (1943), and in Malaya by Orr (1945).

Scott (1955), states that only two epizootics of rinderpest involving sheep or goats have been reported in Africa; one in Egypt in 1863 referred to by Curasson (1932), and an alleged outbreak reported by Koch as quoted by Hutrya and Marek (1922). However, Curasson (1942), states that West African goats may be affected by the natural disease, and refers to the natural disease seen in the Senegal dwarf goat in 1871, and to a small outbreak reported by Claverie in 1927, causing the deaths of 60 goats in French Guinea, no sheep being affected. He makes further reference to the natural disease being observed in imported crossbred goats in the Gold Coast; the local goats were unaffected by contact with these goats. Although Curasson (1942), in discussing sheep and goats as carriers of rinderpest, implies that natural disease in these animals may occur in certain areas of Nigeria, no records of natural rinderpest in sheep or goats in Nigeria can be traced. The disease of sheep and goats discussed by Mornet, Orue, Gilbert, Thiéry, and Mamadou Sow (1956), termed "Peste des petits ruminants," appears to be caused by a virus entity which has antigenic relationship to the rinderpest virus, and causes heavy losses among sheep and goats without affecting contact cattle. These authors are making further investigations into the nature of the virus concerned in order to determine whether it is a variant of the wild rinderpest virus, lapinised rinderpest virus, the goat-passaged rinderpest virus, or some other entity, and until these findings are published the disease cannot be referred to as rinderpest.

An outbreak of rinderpest involving cattle and sheep in the Plateau area of Northern Nigeria is now described, the disease being first observed in cattle, and then apparently spreading to contact sheep. The syndrome observed in the sheep was very similar to the typical rinderpest seen in the cattle, and the disease was confirmed in both cattle and sheep by complement fixation techniques.

History

Thirty-four adult male White Fulani cattle, which were believed to be susceptible to rinderpest, were purchased on July 1st, 1957. At this time of year the pasture was at its most luxuriant, as it was the middle of the wet season, with a mean daily rainfall

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of 0.56 inches, and a temperature variation from 50° to 75° F. No supplementary rations were fed to any of these cattle.

In intimate contact were 110 sheep; an indigenous breed referred to locally as Plateau dwarfs, which are long-haired black and whites, with long, thin tails. The sheep each received 1 lb. per day of supplementary ration consisting of guinea corn 50 per cent., maize 25 per cent., ground nut cake 25 per cent., and to this was added a mineral mixture at the rate of 3 lb. per 100 lb. The sheep were brought from the paddock and housed each night.

On July 15th there was evidence of rinderpest among the cattle; one animal was found dead and 22 became clinically affected, 7 to advanced stages of the disease. Serological confirmation of rinderpest was obtained on July 17th. Control consisted of vaccination, using lapinised rinderpest virus, of 65 susceptible laboratory cattle contacts on July 15th, and slaughter of the infected batch, together with immediate in-contacts on July 17th. No further cases were seen in the contact laboratory cattle until August 16th and 19th when 2 animals which had escaped vaccination became infected. As a precaution, all laboratory cattle received a boosting dose of dried goat virus on August 17th.

Meantime, rinderpest was diagnosed clinically, and confirmed serologically on July 23rd and 24th in the laboratory flock of sheep. Between July 23rd and 25th 54 sheep showed clinical symptoms, and of these, 12 died during this period. Control of the disease in the sheep was attempted by slaughter of those showing clinical symptoms on July 25th, and vaccination of the remainder using lapinised rinderpest virus on the same date. During the next 10 days, 1 of the vaccinated sheep died and 10 more showed clinical symptoms of rinderpest, so the remainder of the flock were slaughtered on August 3rd. Of the slaughtered vaccinated sheep, 3 showed regressing lesions of rinderpest at *post-mortem*. This slaughter policy was necessary as vaccines are produced at Vom, and the risk of contaminating cattle vaccines with a virulent rinderpest virus was very real.

Clinical and *Post-mortem* Findings

The clinical and *post-mortem* findings in cattle were those generally described for rinderpest, with the exception that the typical transverse or "Zebra" markings in the lower bowel, referred to by Henning (1949), were not seen in any of the cattle affected with natural rinderpest, but strangely enough were constant in 4 animals which were diagnosed clinically, serologically, and at *post-mortem* as adverse reactors to the dried goat virus inoculations. The outstanding and most constant lesions in the naturally affected cattle were parallel longitudinal streaks in the lower bowel due to inflammation of the crests of the mucosal ridges. In descending order of frequency and severity, bowel lesions were evident in the first 30 inches of the colon, the caecum, duodenum, abomasum, rectum, occasionally in the ileum, constantly as a yellowish, catarrhal exudate in the jejunum, and none in the first three stomachs. It is of interest to note that longitudinal parallel streaking of the colon is also seen in heartwater cases, but unassociated with erosion or ulceration.

The syndrome in sheep was very similar to that seen in the cattle. As in cattle, the course of the disease in sheep could be roughly divided into the stages described below. No precise data on temperatures can be given, as not only do temperatures fluctuate with time of day, but also the normal temperature of sheep in the tropics is difficult to estimate.

Initial Pyrexia Stage

Affected sheep showed a rise in temperature, with anorexia and retarded rumination, but otherwise it was not easy on visual examination to pick out such animals from a large flock, as they remained bright and active.

Post-mortem findings were not outstanding or distinctive.

Early Dysentery Stage

The temperature was usually lowered, and by the grinding of the teeth and arching of the back severely affected animals showed evidence of abdominal pain, passing mucus and blood which was seldom admixed with faeces. Animals in this and the next two stages could be picked out regularly by the ease with which the thermometer entered a dilated anus. A serous nasal discharge was constantly evident.

Sheep slaughtered in this stage showed a widespread congestion which was particularly evident in the nasal septa, turbinates, pharynx, larynx, retropharyngeal lymph glands, abomasum, duodenum, and lower bowel. As in the cattle, the most constant and characteristic lesions were seen in the lower bowel, evident in every case as a bright cherry-red inflammation of the crests of the mucosal ridges in the caecum, colon, and rectum. Haemorrhage occurred into the lumen of the caecum and upper colon, and the lesions in the colon and rectum were seen as red, parallel, longitudinal, and confluent streaks. The capsule of the spleen was injected, but there was no splenomegaly.

Late Dysentery Stage

The hind quarters and tail were soiled with a black, watery diarrhoea, with occasional streaks of blood and mucus. There was twitching and shivering of the muscles, and frequent coughing. Nasal discharges became either sero-mucoid or muco-purulent. The temperature varied in individual animals, but was usually elevated. Despite these clinical findings sheep in this stage were usually bright and agile, and rarely showed signs of weakness or collapse.

Sheep slaughtered in this stage showed a *post-mortem* picture very similar to the preceding stage, but petechial haemorrhages or commencing erosion were usually evident on the soft palate as opposed to the congestion of the preceding stage. Patchy congestion and emphysema of the lungs was sometimes present, with a regular tracheitis evident as transverse congestion at the junctions of the cartilage rings. Gastro-intestinal lesions were distributed as before, but the intense cherry-red colour of the

longitudinal striping was no longer as striking. Congestion and/or early erosion was sometimes evident in the rectum and colon between these longitudinal stripes, but in no case did they appear as transverse bands of congestion or "Zebra" markings.

Early Diarrhoea Stage

Temperature was usually subnormal, and there was frequent passage of a watery brown diarrhoea soiling the hind quarters. Most of the animals in this stage showed depression and lethargy, although 7 of 19 animals were still bright and active and showed returning appetite. It appeared that, depending on the severity of the infection, animals in this stage could either pass into the last stage, collapse, and die, or if still bright, could show signs of recovery (as in the 3 cases showing regressing lesions noted in the history of the outbreak). The nasal discharge was now frankly muco-purulent; this and the diarrhoea were the most constant clinical features in affected sheep.

Post-mortem findings showed congestion and erosion of the soft palate and the tonsillar tissue frequently contained a greenish-yellow pus. Congestion of the nasal septa and turbinates was still evident, but pharyngitis and laryngitis were far less frequent. Patchy congestion of the lungs, and a tracheitis were regular findings. Abomasal lesions varied from a mild congestion to intense general congestion of the mucosa, with petechiae, ecchymoses, erosions, or ulcerations. Duodenal lesions were very frequent, showing thickening, congestion, and often erosion. Jejunal lesions were still confined to a yellowish, catarrhal exudate; congestion or erosion, when present in the ileum, was confined to its terminal portion. The caecum showed fading congestion with occasional erosion, the most constant lesions still being evident in the first 18 inches of the colon which was thickened, and showed the confluent longitudinal striping, now turning black, to which reference has already been made. The longitudinal striping was also evident at times in the caecum and/or rectum, both of which also showed multiple minute erosions.

Late Diarrhoea Stage

Animals that reached this stage were usually comatose, and died within 24 hours. In general, the clinical picture was as for the previous stage, but the visible membranes were anaemic, the eyeballs sunken, and the nasal discharge was caked and crusted round the nostrils. Coughing was still a feature.

Post-mortem findings were in general as for the previous stage; the longitudinal striping being blackened, but still often confluent in the first part of the colon. Circumscribed consolidations of the tips of the apical and cardiac lobes of the lungs were seen in 3 of 9 cases in this stage, and none of these 3 pneumonias could be accounted for bacteriologically. It should be noted at this point that of 83 *post-mortem* examinations carried out, 16 showed consolidation and necrosis, principally of the apical lobes, but these lesions were not considered part of the rinderpest syndrome on the following counts:—

(a) The advanced state of the lesions and the fact that the degree of necrosis bore no relation to the stage of rinderpest in which affected sheep were slaughtered.

(b) Similar lesions had been observed in laboratory sheep prior to this outbreak, and during the outbreak sheep clinically and serologically negative to rinderpest showed similar lesions.

(c) A micro-aerophilic Fusiformis was regularly isolated from such lesions except in one interesting case in which a pure culture of *Aspergillus fumigatus* was isolated.

A point of some further interest was that all sheep affected with rinderpest showed massive infestation with *Oesophogostomum* nodules, evident as the so-called "pimply gut." In most of these cases, the infection was most evident in the colon, but some cases also showed the caseous necrotic nodules in the small intestine and duodenum. It was noticeable that congestion was well marked round these lesions in the lower bowel.

It will be seen that the syndrome in sheep is similar to that seen in cattle, the following main points of difference being noted:

Salivation

This was not a feature of the disease in sheep, and in only one case were marked mouth lesions present involving the lips and gums. In this case frothing more than salivation was the sign. Mouth lesions in sheep were otherwise confined to the soft palate as petechial haemorrhages leading to erosion, and as abscess formation in the tonsillar tissue.

Ocular Discharges

These were uncommon in the sick sheep, being evident in only 11 of the 65 affected animals, and then not as pronounced as in cattle.

Gall-bladder Lesions

The petechiation and erosion of the gall-bladder lining seen in cattle, was not a feature of the disease in sheep, nor was the thinning of the bile, nor distension of the gall-bladder.

Intestinal Lesions

These were, in general, similar to those seen in cattle; minor points of difference were the more frequent presence of lesions in the terminal portion of the ileum and the typical lower bowel longitudinal striping which tended to blacken and stay confluent in the later stages of the disease instead of breaking up and disappearing as in the cattle.

Course of the Disease

It was difficult to compare the course and severity of the disease in sheep and cattle due to the short time during which they were observed, and the slaughter policy which was adopted. During the 50 hours that they were under observation, the number of sheep showing clinical symptoms increased from 30 to 54, all stages of the disease being well represented. Twelve animals died. From a rough analysis

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of the observations on these 54 animals, supplemented by the findings on the 11 vaccinated sheep which later showed symptoms, a tentative conclusion was that the course of the disease in those sheep that died, or were to die, would be approximately 5 days, each stage lasting 24 hours. Thus, in such sheep the disease was more rapid and severe, compared to the cattle. However, as already noted, the disease was not invariably fatal in sheep, some being affected only mildly and showing evidence of recovery.

Serology

Prescapular lymph glands were collected from sheep and cattle, and tested for the presence of complement-fixing antigen, using as antibody Japanese "O" hyperimmune rinderpest serum. In parallel, serum was collected from sheep and tested for the presence of complement-fixing antibodies, using as antigen Japanese "O" rinderpest virus. The results of this investigation are detailed by Nakamura and MacLeod (1957). In general, antigen or antibody was demonstrable in a proportion of the sheep, and in one case antigen and antibody were present simultaneously. As in cattle, Nakamura (1937), antigen was demonstrable in the early stages of the disease, disappearing sharply in the later stages of the disease as the antibody level rose.

Discussion

The behaviour of the rinderpest virus in sheep and goats presents an intriguing problem. The incidence of the natural disease is low; the syndrome observed in natural and artificial infection is inconstant; and the reports on the rinderpest variant concerned differ in regard to its host-range, and virulence.

The low incidence of the natural disease in sheep and goats is particularly puzzling for Nigeria. Here rinderpest has been widespread among the cattle for many years, but, although sheep and goats are generally grazed with these cattle, this is the first time the disease has been reported in the lesser ruminants. Although staff shortages and poor communications could account for the occasional outbreak in sheep and goats being missed or not reported, it is very unlikely, with rinderpest so rife, that no reports would have been received over the past 30 years. Curasson (1932) states that sheep and goats in Africa are affected severely only in exceptional circumstances. In the light of the present outbreak and the possibility of recurrence, it may be well to theorise on these circumstances, as concerning sheep.

The breed of sheep concerned is known to affect its susceptibility to artificial infection (Nicolle et Adil Bey, 1899; Nocard et Leclainche, 1902; Nencki, 1894; Lingard, as quoted by Curasson, 1942; Lall, 1947). This factor, however, was not involved in this outbreak, since the combination of White Fulani cattle and Plateau dwarf sheep is a common one in Nigeria. Similarly the temperature, humidity, and elevation (4,000 feet) were all common factors to the many surrounding Plateau herds, in which rinderpest confines itself to the cattle.

Two factors, however, which were peculiar to the laboratory sheep infected, were supplementary feeding and fenced pastures. With regard to the first factor, it has already been noted that the sheep concerned were in good condition, the relatively luxuriant grazing being supplemented with concentrates. Thiéry (1956) has observed that rinderpest lesions are more severe in cattle and goats on a high plane of nutrition than in those on a lower plane. The implication of this would be that an inapparent infection occurs in native sheep on a lower plane of nutrition. The second factor peculiar to the sheep affected in this outbreak was that the infected pasture which they grazed was fenced and limited in extent. Native mixed herds are grazed over large, unrestricted areas, and are constantly on the move, suggesting that their intake of infective material is small compared to sheep grazed on a small paddock on which infected cattle had been kept. Curasson (1942) was able to reproduce severe rinderpest in Sudanese sheep only by giving massive doses. This would again presuppose an inapparent infection in sheep receiving small doses. It would be of interest to investigate the incidence of naturally occurring rinderpest antibody in native sheep.

A further point which could have some bearing on the degree of infection in sheep, was that all rinderpest-affected sheep showed a heavy infection of Oesophogostomum, evident as caseo-necrotic, submucosal nodules in the lower bowel, small intestine, and occasionally in the duodenum. Yersin (1901), Walker as cited by Jackson and Cabot (1930), and Plowright (1952) show that sheep may respond to artificial infection with only a viraemia and, occasionally, slight nasal discharge. Assuming the predilection site of the virus to be the alimentary canal mucosa, it would appear that sheep show such an abortive response due to failure of the virus to invade and/or multiply in the intact intestinal mucosa, and that damage to the mucosa may interfere with this protective function. Lastly, of course, one may make the assumption that the wild rinderpest virus is capable, under certain circumstances of mutation in respect to host-range and/or invasiveness, such a mutation having occurred in this outbreak. Bearing on this, it is of interest to note that rinderpest in sheep and goats is believed to be on the increase in India since the introduction of goat-adapted rinderpest virus, used as a vaccine (Crawford, 1947; Daubney, 1949), and that such goat-adapted virus may produce a severe infection in sheep (Stirling, 1933; Skrikantaiah, 1934). It is possible that passage through sheep, similarly, on occasion may induce such a mutation.

Differences in syndrome in response to artificial infection of sheep with the wild virus are evident from a perusal of the literature. Thus Ostertag (1916) was unable to infect sheep; Plowright (1952) described a viraemia and nasal discharge; Edwards (1927) reported a pulmonary form of the disease; Memmo, Martiaglo, and Andani (1904) described a chronic irregular and fatal form, and Curasson (1942) a severe fatal syndrome. The natural disease, however, appears to present a fairly constant syndrome, the outbreaks reported by Bawa (1940) show-

ing a very similar picture to that described in this report; in both cases the disease resembled bovine rinderpest. The variable results obtained by needle passage can in general be attributed to breed and individual susceptibility, to unnatural routes of infection, to different dosages given, or to varying degrees of natural acquired immunity, this depending on the history and husbandry of the sheep used by the various authors, and the incidence of rinderpest in their respective territories. The uniformity of syndrome in naturally infected sheep could similarly be associated with the low incidence and localised foci of the disease, suggesting that unless some precipitating factor is present, as already postulated, the disease may present itself in some mild or irregular form, not suggestive of rinderpest. A suggested line of approach would be to infect sheep by mouth with sheep-passaged virus, using graded doses in animals which are serologically negative to rinderpest.

The importance of clinical rinderpest in sheep is obvious in connexion with the epizootology of the disease in cattle. Although Plowright (1952) states that it is generally conceded that sheep-to-sheep transmission does not occur, and Bawa (1940) and Lall (1947) refer to the disease in sheep as not affecting in-contact cattle, other reports suggest that sheep-to-sheep, and sheep-to-cattle infection does occur (Plowright, 1952; Memmo *et al.*, 1904; Nainsouta, as quoted by Curasson, 1942). Nakamura (1957) believes that the multiplication and excretion rate in sheep is comparatively low, this being indirectly borne out by the serological results in sheep during this outbreak (Nakamura *et al.*, 1957). This factor, in conjunction with other theories in the preceding discussion, may account for the variable reports on the spread of rinderpest from sheep. It is appreciated that the work reported here is unfinished, much of the discussion being pure hypothesis, but in view of the paucity of literature on natural rinderpest in sheep, and the contention of Crawford (1947), and Daubney (1949) that it is on the increase, an interim report was considered justifiable. Future work, tentatively scheduled when isolation facilities become available, is hoped to include comparative studies on the sheep and bovine virus, a comparison of the Nigerian and Indian sheep virus; contact and transmission experiments; and a survey of native sheep sera for the presence of complement-fixing antibodies to rinderpest.

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FIFTY YEARS AGO

The following is an extract from the leading article which was published on May 30th, 1908:—

BUTCHERS *versus* FARMERS

"That the Briton is a law-abiding, long-suffering person is proverbial, and we should think that of all classes of Britons the butcher is the most long-suffering. Some years ago tuberculosis in cattle was not looked upon with any great horror. Unless the patient was very bad, it was killed and eaten with simple faith or ignorance. Then science pointed out that it was dangerous, and Public Health Acts provided for the seizure of meat which was visibly tuberculous. Butchers were prosecuted and fined, not only for selling such meat but for having it in their possession. In very many cases the condition of the animal was not apparent during life, and the butcher paid a good price for a diseased bullock only to have the carcase seized and to lose what he paid for it. In no case does the farmer offer to pay half the loss, and in most districts the farmers decline even to subscribe to an indemnity fund."

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Current Literature

ABSTRACTS

Equine Ringworm with Special Reference to *Trichophyton Equinum*. GEORG, L. K., KAPLAN, W., and CAMP, LA V. B. (1957). *Am. J. Vet. Res.* **18.** 798.

In the past there has been a tendency to name dermatophyte species according to the animal from which it was recovered. Variously labelled Trichophytons were later identified as *T. mentagrophytes*. The authors provide evidence to show that *T. equinum* is a distinct species and that among other cultural characteristics it requires nicotinic acid for effective growth. Ringworm material was obtained from horses in U.S.A. and Canada, Drs. Austwick and Ainsworth provided material from horses in various parts of Britain, and cultures labelled *T. equinum* were obtained from culture collections in different parts of the world.

T. equinum was found to be a common cause of ringworm in horses in U.S.A., Europe, and South America, but some of the cultures made from material from horses and identified as *T. equinum*, and some of the stock cultures from culture collections were identified as *M. canis, T. mentagrophytes* var. granular, *T. verrucosum*, and *M. gypseum*.

Symptoms produced by all types of ringworm are first the appearance of plaques on shoulders, girth and saddle areas, and hind quarters. The plaques are first only perceptible by touch but later become visible as hairs become matted. Slight traction detaches most infected hairs *en bloc* with crust. Plaques are first moist and later become dry, powdery, and slate grey in colour. There is often severe itching. S. J.

Intracerebral Inoculation of Distemperoid Virus: 1.—Technique and Possible Indications in Canine Encephalitis. LUTSKY, I. (1957). *N. Amer. Vet.* **38.** 339-43.

A new method of treatment for encephalitis following distemper is being investigated by the author, and in this first report the method whereby a 75-mg. dose of ferret modified canine distemper virus (Distemperoid) is injected intracerebrally is described.

Under general anaesthesia a ¼-inch midline incision of the skin covering the skull is made at the level of the frontoparietal suture. The skin is reflected laterally and the skull is penetrated with a sterile drill or awl. A hypodermic needle is inserted alongside the drill or awl which is then withdrawn. The hypodermic needle is inserted fully into the parietal cortex at an angle of 45 degrees to the median plane, and the vaccine injected slowly. The needle is withdrawn and the skin is resutured into its normal position.

By this somewhat drastic means it is possible to protect those brain cells which have not yet been affected by the virulent distemper virus, such protection being effected by viral interference. The author also claims that the intracerebral injection of Distemperoid prior to the appearance of nervous symptoms, has been effective in preventing the onset of encephalitis.

While vaccine therapy by this technique holds out interesting possibilities for further investigation it is felt that the claim for effective prophylaxis would have to be proved over a large number of cases using adequate controls, before such a drastic method of treatment could be universally adopted. J. S. J. L.

The Influence of Maturity and Sex on the Blood Pressure of the Turkey. WEISS, H., & SHEHAN, M. (1958). *Am. J. Vet. Res.* **19.** 209-11.

The authors stated that there was no difference between the blood pressures of male and female poults at 6 to 7 weeks of age. Turkeys 8 to 9 months old showed a higher systolic pressure and a lower heart rate than the poults, the pulse pressure of the males being significantly higher than that of the females.

It is suggested that the blood pressure pattern of the adult males may be associated with the presence of atheromatous lesions of the aorta, and that the elevated pressures may, in turn, lead to aneurysms and rupture of the defective vessels. This would help to explain the higher incidence of aortic rupture in male birds. E. A. G.

The Effect of Divided Dosage on the Anthelmintic Efficiency of Phenothiazine in Lambs. DOUGLAS, J. R., *et al.* (1957). *J.A.V.M.A.* **130.** 257-9.

Groups of 40-lb. lambs carrying mixed infestations of Ostertagia, Trichostrongylus, and Nematodirus species received a 25-g. dose of phenothiazine. The dose was administered as a single dose or divided equally over 3, 5, and 7 days. Anthelmintic efficiency was assessed on worm counts of stomach and intestinal contents carried out at *post-mortem* examination 7 days after treatment ended. Anthelmintic efficiency decreased from 65 per cent. for the undivided dose to 46 per cent., 32 per cent., and 0 per cent., respectively, when the dose was divided into equal portions administered over 3, 5, and 7 days.

The authors conclude that recommendations for the division of a phenothiazine dose cannot be supported if maximum therapeutic efficiency is to be attained. W. B. S.

Food Poisoning in England and Wales, 1956. A Report of the Public Health Laboratory Service. *Mon. Bull. Minist. Hlth. Lab. Serv.* (1957). **16.** 233-41.

The overall picture was similar to that for previous years. Of the 7,713 incidents reported, 41 per cent. were due to *Salmonella typhi-murium* infection and 15 per cent. to other Salmonella organisms. The cause remained undiscovered in a further 41 per cent. of the incidents. Thirty-five of the 44 fatal cases were due to Salmonella infections and five to *Staph. aureus*. Four outbreaks were associated with milk, but in only one—due to *S. typhi-murium*—was the infection traced back to cattle. E. A. G.

News and Comment

THE COUNCIL OF THE R.C.V.S.

The Minister for Agriculture for the Republic of Ireland has reappointed Professor J. A. Nicholson, M.A., PH.D., M.V.SC., M.R.C.V.S., Principal of the Veterinary College of Ireland, to be one of the Irish representatives on the Council of the Royal College.

Professor P. E. Mullaney, M.A., M.V.M., M.R.C.V.S., Professor of Pathology at the Veterinary College of Ireland, has been re-elected as the General Elected Member for the Republic of Ireland on the Council of the Royal College.

Both Professor Nicholson and Professor Mullaney will hold office until the Annual General Meeting of the College in 1962.

PERSONAL

Birth

DAVIES.—On May 20th, 1958, to Catherine, wife of Michael G. Davies, M.R.C.V.S., of Dragonscroft, Westwood Road, Leek, Staffs, a son.

Marriage

SOUTHERTON—HERRIDGE.—On May 17th, 1958, at Saint Michael's Church, Shrewsbury, Anthony George Southerton, B.VET.MED., M.R.C.V.S., N.D.A., 8, Denmark Terrace, Shifnal, Salop, to Margaret Virginia Herridge.

IN PARLIAMENT

Irish Cattle (Bovine Tuberculosis)

MR. HURD (May 22nd) asked the Minister of Agriculture, Fisheries and Food what progress has been made in talks with the Irish authorities on the safeguards to be applied when Irish cattle are brought into attested areas free from bovine tuberculosis in Great Britain.

MR. GODBER: Representatives of my Ministry took part in discussions at the Department of Agriculture in Dublin last week on future arrangements for imports of Irish cattle into Great Britain in the light of progress with the eradication of bovine tuberculosis in both countries. My right hon. Friend the Secretary of State for Scotland and my right hon. Friend are considering the reports of these discussions, but I am not at present in a position to make a statement.

Pigs (Atrophic Rhinitis)

MR. DYE (May 22nd) asked the Minister of Agriculture, Fisheries and Food (1) how many cases of atrophic rhinitis have occurred in Norfolk in the past two years; how quickly steps are taken to isolate or destroy any pigs so affected; and what advice he gives to owners of pigs who suspect their animals are suffering from this disease;

(2) how many cases of atrophic rhinitis amongst pigs have occurred in the United Kingdom during 1957, and up to the latest available date in 1958; and what steps are being taken to eradicate this disease.

MR. GODBER: During the two years ended May 20th, 1958, five cases of atrophic rhinitis occurred in Norfolk. In the United Kingdom 17 cases were investigated in 1957 and seven up to May 20th, 1958. Under the Atrophic Rhinitis Order, 1954, when a veterinary inspector suspects the presence of the disease, the pigs concerned are detained and isolated to prevent the spread of infection. Further investigations, including *post-mortem* examinations, then take place. If the presence of the disease is confirmed all the affected pigs are slaughtered with payment of compensation; and if there is evidence of spread in the herd, any pigs which may have been exposed to infection may also be slaughtered. Owners and persons in charge of pigs are required by the Order to report to the police the existence or suspected existence of the disease. There is also provision for disinfection of premises and articles that may be contaminated where an outbreak has occurred. My right hon. Friend is making inquiries about the complaint of which the hon. Member has sent him particulars and he will write to him.

Rabbit Clearance (Advisory Council)

MR. CHAMPION (May 22nd) asked the Minister of Agriculture, Fisheries and Food why no veterinary surgeon has so far been included amongst the 14 members of the Advisory Council on Rabbit Clearance; and whether, in view of the desirability of having expert guidance available for this body, he will either appoint a veterinary expert as a member of the Council or arrange for one to be appointed as a technical consultant.

MR. GODBER: The Council has arranged for my Ministry's chief veterinary officer to attend its meetings, when necessary, as an assessor and also to act as chairman of its Scientific Sub-Committee, which includes four other veterinary surgeons.

Composition of Milk (Committee)

MR. J. LINDSAY (May 22nd) asked the Minister of Agriculture, Fisheries and Food whether he will now announce the terms of reference and the membership of the proposed committee on the composition of milk.

MR. JOHN HARE: The Committee's terms of reference will be as follows:—

"To consider the composition of milk sold off farms in the United Kingdom from the standpoint both of human nutrition and of animal husbandry and to recommend any legislative or other changes that may be desirable."

The Chairman will be Dr. J. W. Cook, F.R.S., D.SC., PH.D., F.R.I.C., Vice-Chancellor of Exeter University, and the following have agreed to serve as members of the Committee:—

Professor R. G. Baskett, O.B.E., M.SC., F.R.I.C.; S. Clifford, Esq., C.B.E., A.C.G.I., B.SC. (ENG.), A.M.I.M.E.; Miss R. L. Cohen, M.A.; Professor E. L. Crossley, B.SC., F.R.I.C., F.L.S.; D. P. Cuthbertson, Esq., C.B.E., M.D., D.SC., F.R.S.E.; P. R. Evans, Esq., M.SC., M.B., CH.B., M.D., F.R.C.P.; G. N. Gould, Esq., J.P., M.R.C.V.S.; Professor W. Holmes, PH.D., B.SC.; H. E. Magee, Esq., M.B., B.CH., M.R.C.P.; Professor B. S. Platt, C.M.G., M.B., CH.B., M.SC., PH.D.; A. Robertson, Esq., D.SC.; R. Waite, Esq., M.SC., PH.D., A.R.I.C.; H. L. Webb, Esq., M.A.; E. C. Wood, Esq., PH.D., A.R.C.S., F.R.I.C.

COMING EVENTS

June

4th (Wed.). 16th Annual General Meeting of the Society of Women Veterinary Surgeons at the Meeting Hall of the Zoological Society of London, Regent's Park, N.W.1, 2.30 p.m.

General Meeting of the South Wales Division at the Cambrian Hotel, Saundersfoot, 2.30 p.m.

5th (Thurs.). Annual General Meeting of the Shropshire Veterinary Club at the Oak Hotel, Shelton, Shrewsbury, 8 p.m.

6th (Fri.). Annual Dinner and Reunion of the Royal Veterinary College Association at the Royal Veterinary College, Camden Town, 6.30 for 7 p.m.

Summer Meeting of the North of England Veterinary Medical Association in the Forestry Lecture Room, Agricultural Department, King's College, Newcastle upon Tyne, 2.30 p.m.

7th (Sat.). 12th Annual Sports Day of the Students' Union Society at the Goring Cricket Ground, Goring-on-Thames.

11th (Wed.). 115th Annual General Meeting of the Royal College of Veterinary Surgeons at 10, Red Lion Square, London, W.C.1, 3.45 p.m.

Annual General Meeting of the Victoria Veterinary Benevolent Fund at 10, Red Lion Square, W.C.1, 10.15 a.m.

Meeting of the North Wales Division at Bryn Adda, Dolgellau, 2 p.m.

12th (Thurs.). Annual Meeting of the R.A.V.C. Golfing Society at the North Hants Golf Club, Fleet, 10 a.m.

13th (Fri.). Annual Dinner of the Royal Army Veterinary Corps Officers' Club at Grosvenor House, Park Lane, London, W.1.

17th (Tues.). Meeting of the Western Counties Veterinary Association at the Hotel Bristol, Newquay, 2.30 p.m.

20th (Fri.). Meeting of the Society for the Study of Animal Breeding at Lyndhurst, Hants.

Annual General Meeting of the Lincolnshire and District Division at the Eastgate Court Hotel, Lincoln, 2.15 p.m.

25th (Wed.). Summer Meeting of the Herts and Beds Veterinary Society.

September

21st to 26th (Official Opening, Monday, September 22nd). 76th Annual Congress of the British Veterinary Association in Douglas, Isle of Man.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN LONDON

Wednesday, July 9th—No. 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Parliamentary and Public Relations Committee.
2.00 p.m. Finance Sub-committee.
3.45 p.m. Home Appointments Committee.

Thursday, July 10th—No. 7, Mansfield Street

9.30 a.m. Small-Animals Committee.
11.30 a.m. Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, July 11th—Connaught Rooms, Gt. Queen Street, W.C.2

10.15 a.m. Council.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Lancs. Crabtree Farm, Preston Road, Inskip, Preston (May 19).

Fowl Pest

Aberdeen. Braehead, Bainshole, Huntley (May 17).

Cambs. 3, St. John's Road, Ely; 2, Ely Road, Little Thetford, Ely; 5, Ely Road, Little Thetford, Ely (May 16); The Orchards, Gruntey Fen, Wilburton, Ely (May 17); Damhead Farm, Wayhead, Coveney, Ely (May 18); Poultry Houses, Common Road, Witchford, Ely (May 20).

Ches. Beechwood Farm, Kermincham, Holmes Chapel, Crewe (May 19).

Hants. Tanglewood, Grosvenor Road, Soldridge Medstead, Alton (May 21).

Lancs. Heatons Bridge, Scarisbrick, Ormskirk (May 18).

Sussex. Perryfield Pig and Poultry Farm, Small Dole, Henfield; Denfurlong, Shoreham Road, Henfield; Racehill Farm, Brighton (May 21).

Swine Fever

Anglesey. Tyddyn Hirion, Llanynghenedl, Valley (May 21).

Bristol. Elm Farm, Burnett, Keynsham (May 16); The Bungalow, Redfield Hill, Bitton (May 19).

Ches. Green Hayes, Barnston Road, Barnston, Heswall, Wirral (May 16); Meadow View, Crewe Lane, Farndon; Wood View Dairy, Kelsall (May 20).

Dorset. Bellair, Whitchurch Canonicorum, Bridport (May 19).

Flints. Yr Erw, Abergele Road, Rhuddlan, Rhyl (May 19).

Glouc. 11, Aston Magna, Moreton in Marsh (May 20).

Hants. Plantation Farm, Wolverton, Basingstoke (May 20).

Lancs. Fulwood Hall Farm, Fulwood Hall Lane, Fulwood, Preston; Banker Street Piggeries, Bolton (May 20).

Leics. Home Farm, Horninghold, Market Harborough (May 19).

Middx. St. Bernards Hospital, Southall (May 20).

Norfolk. Meadowhurst, North Ipham, Diss (May 16); The Lodge Farm, Breckles, Attleborough (May 19); Allotment Premises, Estuary Road, King's Lynn (May 20).

Shrops. Common Farm, Hatton, Shifnal (May 19).

Somerset. Stawell Fruit Farm, Stawell, Bridgwater (May 19).

Staffs. The Red, White and Blue Inn, Lichfield Road, Springhill, Lichfield (May 19); 23 Holding, Watling Street, Weaford, Lichfield (May 20).

Suffolk. Tye Farm, Alpheton; Church Farm, Finningham Road, Rickinghall Superior, Diss; The Mill, Lavenham Road, Cockfield, Bury St. Edmunds (May 19); Chilton Hall Farm, Bury Road, Stowmarket; Lodge Farm, Coney Weston, Bury St. Edmunds (May 20).

Sussex. Batchelors Farm, Langney, Eastbourne (May 20).

Warwicks. Nursery Pig Farm, Hampton-in-Arden, Solihull (May 16).

Yorks. Manor Farm, Malmerby, Ripon (May 20).